1062-78 Left Ventricular Intracavitary Gradients Are an Underreported Cause of Systolic Murmur in Adults

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This prospective, observational study investigated LV intracavitary gradients in 108 consecutive patients referred to the Tucson VA Medical Center Echocardiographic Laboratory for evaluation of a systolic murmur of unknown origin. Complete two-dimensional echo-Doppler studies were performed. Ventricular intracavitary gradients were demonstrated by color and pulsed-wave Doppler mapping of intracavitary velocities. Clinical data were obtained from patient histories, examinations and medical records. Areas of maximal intensity and characteristics of the murmur were noted.

Left ventricular intracavitary gradients were present in 35.2% of the study population, and were the sole explanation for systolic murmur in 12%. These short murmurs were localized to the left lower sternal border. Additional sources of systolic murmur in decreasing order of incidence were mitral regurgitation (43.5%), tricuspid regurgitation (35.3%), aortic stenosis (26.9%), aortic sclerosis (23.1%), hypertrophic cardiomyopathy (0.9%) and ventricular septal defect (0.9%). Compared to patients without LV intracavitary gradients, patients with LV intracavitary gradients had an increased percentage fractional shortening (44.3 ± 10.0 vs. 31.5 ± 10.5%, $p < 0.001$), LV mass index (187 ± 78 vs. 153 ± 44 g/m^2, $p = 0.019$), and diastolic wall thickness relative to cavity dimension (61.4 ± 16.0 vs. 45.5 ± 12.6%, $p < 0.001$). The group with LV intracavitary gradients had a higher prevalence of hypertension (78.4% vs. 50.7%, $p = 0.011$) and a trend towards a lower prevalence of coronary artery disease (18.9% vs. 37.9%, $p = 0.076$). The two groups were comparable for age (66.2 ± 14.1 vs. 65.2 ± 13.0 years, $p = 0.715$) and proportion of females (13.2% vs. 12.9% females, $p = 0.799$).

In the presence of LV hypertrophy and absence of hypertrophic cardiomyopathy, LV intracavitary gradients are a relatively common cause of systolic murmurs in this Veteran patient population. This previously underreported etiology should be included in the differential diagnosis of systolic murmurs in adults.

1062-79 Differences in Regional Mechanical Function Between Concentric and Eccentric Left Ventricular Hypertrophy: A Study Using Magnetic Resonance Tissue Tagging

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To determine whether there are systematic differences in regional intramural mechanical function between concentric LVH (CH) and eccentric LVH (EH) in patients matched for age, LV mass, and global LV function, we obtained magnetic resonance tissue-tagged images in 14 patients (mean age 50) with CH from systemic hypertension, and 10 patients (mean age 47) with EH due to isolated moderate aortic regurgitation. All patients had normal LVEF and % LV shortening by 2D echo and similar LV mass index (156 ± 56 v. 151 ± 30 g/m, CH v. EH, p = NS). Two-dimensional finite element analysis was used to quantitate the principal orthogonal strains $\lambda 1$ (greatest systolic elongation) and $\lambda 2$ (greatest systolic shortening), β (angular deviation of $\lambda 1$ from the radial direction), and *D* (element displacement) at 3 LV short axis levels in the anterior, lateral, inferior, and septal walls.

Region	$\lambda 1$		β(°)		D (mm)	
	EH	CH	EH	CH	EH	CH
Anterior	0.22	0.12*	11	17*	5.8	5.4
Lateral	0.19	0.13*	11	17*	8.6	5.6*
Inferior	0.21	0.08*	12	21*	8.3	5.9*
Septal	0.20	0.12*	14	23*	4.5#	4.2*

*$p < 0.05$ EH vs CH, #$p < 0.05$ Septal vs other regions

$\lambda 1$ was significantly reduced in all regions in CH compared to EH, while $\lambda 2$ was similar (−0.18 v. −0.17, EH v. CH, p = NS). Element displacement, *D*, was nonuniform in EH, while β was more circumferentially oriented in CH than EH. We conclude that systematic differences in intramural mechanics exist between concentric and eccentric hypertrophy. These findings may relate to differences in LV chamber architecture, fiber orientation, or cross-fiber shortening due to the differing patterns of LV remodeling.

1062-80 Force-Frequency and Relaxation-Frequency Relationships in Hypertrophied Hearts of Children

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The combined effects of chronic pressure-overload hypertrophy and heart rate (HR) on left ventricular (LV) contractility and relaxation in children with congenital aortic stenosis (AS) are poorly understood. We studied 6 children (age: 2–13 years) with AS and LV hypertrophy (H) and 6 controls (C), under sedation. LV mass was calculated by M-mode echocardiography. Maximum rate of rise of LV pressure (dP/dt_{max}) was used as an index of contractility (force) and time constant of LV relaxation (τ) was used as an index of relaxation. These indices were calculated from micromanometric LV pressure tracings at constant HR of 110, 120, 130, 140, 150 and 160 bpm generated by atrial pacing. LV mass normalized for height, was significantly increased in H vs C groups (98.6 ± 15.7 vs 58.6 ± 14.2 g·m^{-1}, $p < 0.05$). Positive force-frequency (dP/dt_{max} vs HR) and relaxation-frequency relationships (τ vs HR) remained intact in both H and C groups indicated by their unchanged slopes (force-frequency 16.4 ± 2.0 (H) vs 16.7 ± 0.9 (C), $p > 0.05$ and relaxation-frequency −0.26 ± −0.06 (H) vs −0.22 ± −0.04 (C), $p > 0.05$). In H group, force-frequency relationship was shifted upwards but mean dP/dt_{max} at each HR was not significantly different (Fig. 1). Relaxation-frequency relationship was shifted upwards and mean τ, in contrast, was significantly prolonged at each heart rate in H group (Fig. 2, *$p < 0.05$), suggesting impaired relaxation.

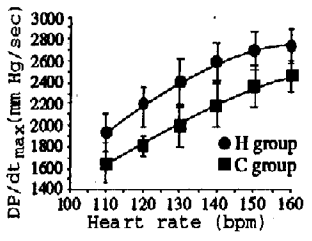

Fig. 1. Force-frequency relationship.

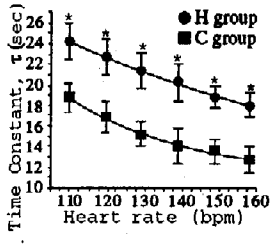

Fig. 2. Relaxation-frequency relationship.

Conclusions: Heart rate has a positive inotropic and lusitropic effect in children with both normal and hypertrophied LV. In hypertrophied LV, force-frequency and the relaxation-frequency relationships remain intact. Although contractility remains unaffected, relaxation is impaired in hypertrophied hearts of children.

1062-81 Early Left Ventricular Mass Regression Measured by 3D Echocardiography in Hypertensive Subjects Is Independent of Reduction in Left Ventricular Volume

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Background: Measurement of the regression of left ventricular (LV) mass is important for assessing the efficacy of antihypertensive therapy. We have previously demonstrated that a reduction in LV mass 6 weeks after initiation of antihypertensive therapy is measurable by three-dimensional echocardiography (3D ECHO), but not by 1D or 2D echocardiography. This occurs because of improved accuracy and reproducibility of 3D ECHO which is not limited by geometric assumptions or image plane positioning error. There is little data, however, to indicate whether mass regression detected early (after 6 weeks of treatment) may be due, at least in part, to overall volumetric changes of the heart or whether it is due only to reduction of muscle mass. *Purpose:* To determine if reduction of LV mass as measured by 3D ECHO after 6 weeks of therapy occurs independently of LV end-diastolic volume (EDV) change. *Methods:* 20 patients with uncontrolled hypertension and hypertrophy underwent 3D ECHO at baseline and after 6 weeks and 12 weeks of therapy. LV chamber volume and mass (myocardial volume) were computed by a 3D ECHO surface reconstruction algorithm using 8–10 short-axis images guided by an acoustic spatial locater and line of intersection display. *Statistical Analysis:* Mean changes of LV mass and volume were analyzed by repeated measures ANOVA using Tukey's test of the means. *Results:* LV EDV showed no significant change after 6 weeks of therapy, whereas mass had undergone significant reduction.

Weeks	n	Mass Diff	p	Vol Diff	p
0–620	11.5 g	0.0018*	6.7 ml	0.114	(ns)

Conclusion: Left ventricular mass regression, as measured by 3D ECHO after 6 weeks of therapy is not attributable to significant changes in left ventricular end-diastolic volume.